



REVIEW ARTICLE

Genetic determinants underlying major disease resistance in sunflower: A review

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Abstract

Sunflower (*Helianthus annuus* L.) is an important oilseed crop worldwide, valued for its high-quality edible oil which is a rich source of unsaturated fatty acids and rich in vitamin E. It is widely preferred due to its adaptability to diverse agro-climatic conditions and short growing period. Statistical reports from Indiastat revealed that the sunflower cultivation in India covers 1.25 % of the global area and contributes around 0.58 % of the world's production. However, its productivity is often hindered by many biotic stresses, especially diseases ranging from 30 % to 70 %. Although pest infestations do occur, diseases are considered more critical due to their rapid spread, persistent nature and potential for severe crop losses across various growing seasons. Considering all the factors, effective disease management is thus essential to sustain and enhance sunflower production, ensuring food security and economic stability for farmers. It is essential to comprehend the genetic factors that influence disease resistance to create cultivars that are high-yielding and durable and to improve crop sustainability along with reduced fungicide need. The promise of developing sunflower cultivars with broad-spectrum and long-lasting resistance lies in the integration of conventional and molecular approaches such as detection of resistant quantitative trait loci (QTLs), utilisation of wild species and the application of genomic selection strategies. This review consolidates existing insights into the genetic keystones of disease resistance in sunflower and highlights how wild *Helianthus* species serve as important reservoirs of resistance genes.

Keywords: diseases; omics; resistant QTLs; sunflower; wild species

Introduction

Sunflower (*Helianthus annuus* L.), is an annual, monoecious, dicotyledonous plant that belongs to the family of *Asteraceae* (1), with chromosome number $2n = 34$ (2) and has an estimated genome size of 3000 Mbp (3). The term "*Helianthus*" has originated from the Greek words "*Helios*," which means sun and "*Anthus*," which signifies flower. The sunflower exhibits an astonishing behaviour of following the sun's movement hence it is called "Girasol" in Spanish and "Tournesol" in French (4). Sunflower ranks fourth among the oil-producing crops after oilpalm, soybean and canola (5). Sunflower is one of the important oil crops and contributes to about 12 % of the global vegetable oils and 10 % of the total edible oil (FAOSTAT, 2018). Its utility can be seen in its farming regions from the sub-tropical to sub-arctic regions amid other areas where it is exploited for the preparation of high-quality consumable oils (6). Sunflower oil has been viewed as a healthy vegetable oil for ages and it is considered premium oil for salad, cooking and margarine production and is also used as a source of biodiesel in recent times. Sunflower is primarily an oil crop, with high protein meal as a by-product. Sunflower pellet production is also important

among the nations, as it serves as the principal grinding sub product. With the increasing demand for oil production next to food production, sunflower plays a vital role in meeting the demand and supply of edible oil and satisfying economic and dietary needs. Sunflower oil is rich in polyunsaturated linoleic acid by nature which contributes about 70 % of the total sunflower oil content and this is followed by monounsaturated oleic acid which makes up about 20 % of total sunflower oil content (7). High-oleic acid content of sunflower oil lays the foundation for higher demand in the market along with health benefits of heart-healthy properties (8).

Sunflower is one of the fastest growing oilseed crop and India ranks second in sunflower production in Asia after China. The total area under sunflower cultivation in the world is about 26.42 million hectares, the production is about 54.45 million tons and the average yield is about 1.93 Mt/ha (USDA 2022-2023). India occupies an area of about 3.64 lakh hectares with the production of 3.63 lakh tonnes (Indiastat 2022-2023). In India, the major sunflower-growing states are Karnataka, Maharashtra, Andhra Pradesh, Tamil Nadu and Gujarat (9).

However, its production is frequently threatened by various fungal, bacterial and viral diseases, which can significantly reduce yield, oil content and overall crop quality. Among various diseases affecting sunflower, most devastating diseases are downy mildew (*Plasmopara halstedii*), powdery mildew (*Golovinomyces cichoracearum*), rust (*Puccinia helianthi*), Sclerotinia head rot (*Sclerotinia sclerotiorum*), Alternaria leaf spot (*Alternaria helianthi*) and Sunflower Necrosis Virus (SFNV).

For an instance Sunflower Necrosis Disease (SND) was noted as an epidemic disease in southern states of India during 1997-1999, with incidence ranged from 10 % to 80 % and yield loss upto 90 %. SND had caused a steep decline in cultivation of sunflower in India from 2.34 million hectares during 2005-06 to less than 2.5 lakh hectares (DOR Annual report, 2001). Sunflower yield is also affected to nearly 50 % by downy mildew, 30-74 % by powdery mildew, nearly 80 % by rust, nearly 60 % by head rot and 27-80 % by leaf spot (4). Hence, knowledge on controlling of sunflower diseases under threshold level becomes a key area of concern.

Managing these diseases effectively requires a combination of cultural practices, chemical control and most importantly, the development of disease-resistant sunflower varieties. Traditional breeding, genetic modification and molecular marker-assisted selection have played a crucial role in enhancing sunflower resistance to major pathogens. Resistance genes (R-genes) have been identified for key diseases such as downy mildew and rust, enabling breeders to develop hybrids

with improved tolerance. This article explores the major diseases affecting sunflower, their impact on production, the genetic basis of resistance and recent advancements in breeding strategies that contribute to improving disease resistance in this economically important crop.

Major diseases of sunflower and their impacts

Major diseases that affect sunflower and their causal organisms are listed in Table 1. Mode of spread of disease pathogens can be broadly classified into soil, air and vector borne. Different diseases are found to be spread by various modes which are mentioned in Fig. 1.

Downy mildew

Symptoms

The most severe symptoms in the field are caused by root infections in immature sunflower plants, which can significantly affect yield (10). Damping off of sunflower seedlings, short stature of plants, leaf bleaching from veins and observable white sporulation starting on the underside of cotyledons and leaves are some of the other major symptoms (11).

Effects and losses

Sunflower downy mildew, which is spreading in all major sunflower-growing countries of the world except Australia, has been considered to be the worst foliar disease that ever occurred (12). It often damages nearly 50 % of the plants and is responsible for more than 80 % loss of potential production (13).

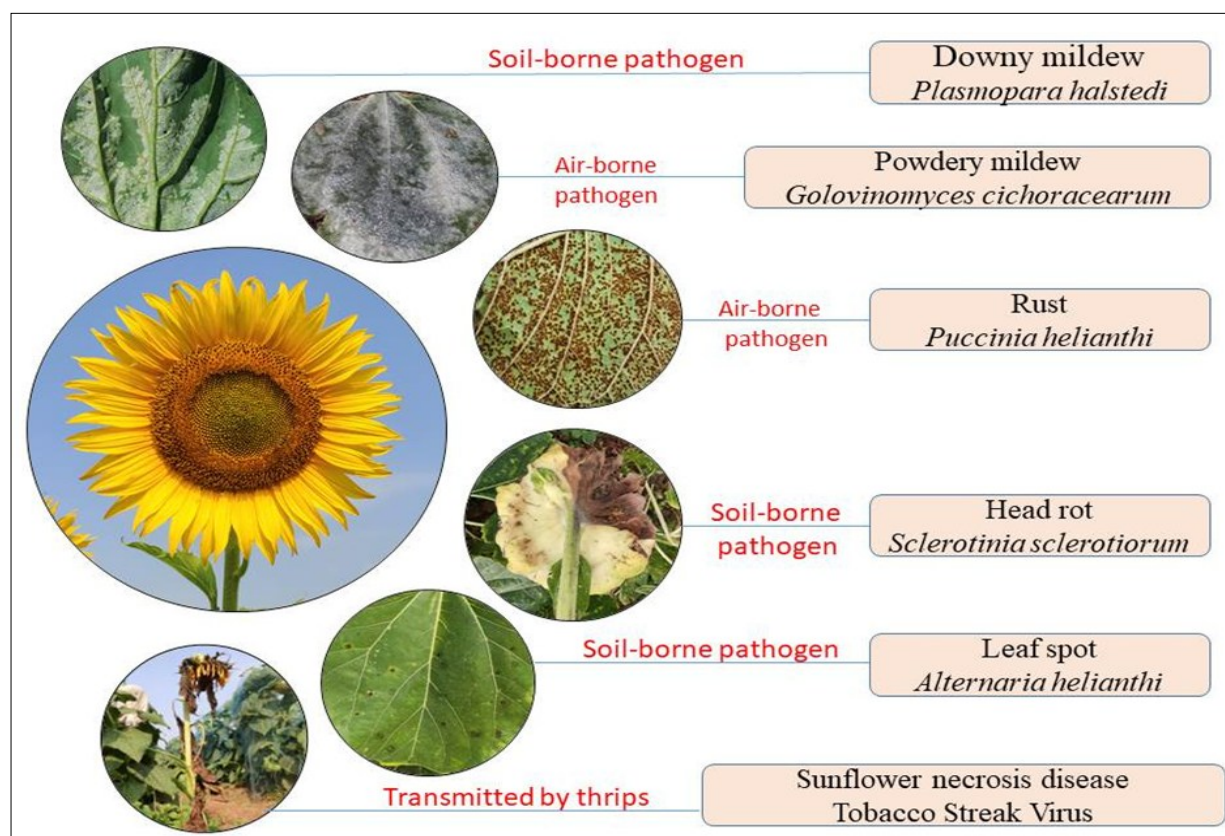


Fig. 1. Major diseases of sunflower with their causal organism and mode of spread.

Table 1. Major diseases of sunflower and their causal organism

Sl.No	Disease	Causal organism
1.	Downy Mildew	<i>Plasmopara halstedii</i>
2.	Powdery Mildew	<i>Golovinomyces cichoracearum</i>
3.	Rust	<i>Puccinia helianthi</i>
4.	Head Rot	<i>Sclerotinia sclerotiorum</i>
5.	Leaf Spot	<i>Alternaria helianthi</i>
6.	Sunflower Necrosis Disease	Tobacco Streak Virus

Disease cycle

The life cycle begins with overwintering oospores in the soil. In spring, these oospores germinate to produce zoosporangia, which release motile biflagellate zoospores into free water in the soil (14). Upon contacting sunflower roots, zoospores encyst and germinate within a few hours, infecting roots either by directly penetrating root cells (sometimes through the formation of appressoria or by entering through wounds), especially at the base of root hairs. Inside the root, the pathogen forms infection vesicles and spreads through the intercellular spaces of the cortex to colonize shoot tissues, where it develops haustoria to extract nutrients (11). During humid and moderately warm conditions in late spring and summer, the pathogen emerges on the undersides of leaves and cotyledons through stomata and produces branched zoosporangiophores that bear zoosporangia (10). These zoosporangia can disperse to nearby plants, releasing zoospores that encyst and penetrate leaf tissues, mainly at cell junctions using appressoria and more rarely through stomata. The progression of the pathogen within infected leaves is not yet fully understood. Secondary infections, which occur under cool and humid conditions, typically result in milder symptoms than those caused by primary root infections (11).

Disease diversity

Downy mildew pathogen was originally classified into two groups: the European race, considered the least virulent and the more aggressive North American or Red River Valley race (15). These were later designated as Race 1 and Race 2, respectively and were initially believed to be limited to their respective continents. In 1980, a third race emerged in the U.S., described and was sometimes called the “new” Red River Valley race (16). Subsequent discoveries included Race 4 in 1984 (17) and a highly virulent Race 5 in 1987 (18), both seemingly restricted to North America. By using a set of 12 public differential lines, researchers had identified at least 12 distinct races or virulence patterns by early 1996 (19). Interestingly, Races 4 and 5 were also detected in Germany (20). In Hungary, six races of *Plasmopara halstedii* had been identified, including Races 2, 4, 8 and 9. Globally, Race 1 is typically the most widespread. In the Indian state of Maharashtra, Race 1 has been reported (21).

Control measures

Choice of planting sites and disposal of infected crop residues also give a fairly good control. Maintaining a minimum four-year crop rotation is essential because the oospores remain viable for many years in the soil. Effective seed treatments include oxadixyl and benalaxyl, analogs of metalaxyl and to a lesser extent, acetamide and triadiazole compounds. Seed treatment with metalaxyl at the rate of 3 g per kg of seed has been found to give effective control (22). While metalaxyl completely suppresses aboveground symptoms and fungal sporulation, *P. halstedii* is still able to colonize portions of the seedling root. Metalaxyl applied as a soil drench was effective as an eradicator, but the rates would similarly be cost prohibitive for large-scale field applications. Both mycoparasitic fungi such as *Trichoderma* spp., *Gliocladium* spp. and *Pythium* spp. and bacteria, such as *Bacillus* spp., *Enterobacter* spp. and *Pseudomonas* spp., have been shown to control diseases caused by various *Pythium* spp. and *Phytophthora* spp. (14).

Powdery mildew

Symptoms

The symptoms of powdery mildew by *Erysiphe* and other genera during the early stages of the crop are little, dull-white, spherical spots on the leaf surfaces which later turn into black spots, or cleistothecia, may start to show up on the leaf toward the conclusion of the growing season. Eventually, white mycelium will cover the leaves. Larger, blended white to grey spots cover much of the plant. As the season progresses, the powdery, hazy mildew leaves turn brown. It has been shown that when leaves ripen, the infection shifts from the more infected, lower leaves to the more diseased, upper leaves. These patches of white fungal mycelium that are easily removed from both the upper and lower leaf surfaces (23).

Effects and losses

Over the past ten years, powdery mildew has become a serious issue and has quickly spread to all of India's sunflower-growing regions (15, 16). Although powdery mildew is found all over the world, depending on the severity of the disease, higher intensity is observed on sunflower in tropical regions and 30-74 % in India. One of the main issues with sunflower farming in the tropics and subtropics nowadays is powdery mildew (23, 24).

Disease cycle

The fungus *Golovinomyces cichoracearum* causes powdery mildew in sunflower and survives between seasons as conidia or cleistothecia (resting spores) on plant debris or alternative hosts. In favourable conditions - typically warm, dry days with cool, humid nights, the airborne conidia are released and land on sunflower surfaces, where they germinate without the need for free water (23). The fungus forms a superficial mycelium on the leaf surface and allows haustoria into epidermal cells to draw nutrients. It continues to reproduce asexually by producing more conidia, leading to rapid spread across the crop. The disease appears as white, powdery spots on leaves, stems and eventually flower parts, reducing photosynthesis and yield (24).

Control measures

Powdery mildew is controlled by applying protective fungicides such as sulfur, triazoles, or strobilurins at the early onset of symptoms. Avoiding excessive nitrogen fertilization can reduce the density of susceptible plant tissues. Additionally, ensuring adequate spacing between plants promotes airflow and reduces humidity, which can help suppress fungal development (22).

Rust

Symptoms

Rust symptoms include reddish-brown pustules that can merge and significantly reduce plant productivity. With a few notable exceptions, rust was frequently more common than 50 % in the locations under study and in some years, it was much more common (4).

Effects and losses

In a year with a rust pandemic, yield losses can reach 80 % when the atmosphere is conducive to the growth of rust diseases (4). One of the rare illnesses frequently found on sunflower is sunflower rust.

Disease cycle

Puccinia helianthi, the rust pathogen of sunflower, is an autoecious fungus that completes its life cycle on sunflower and overwinters as teliospores on crop residue. In spring, teliospores germinate to form basidiospores, which infect sunflower tissues and produce pycnia (yellow-orange humps on upper leaves) and aecia (orange cup-like structures on the underside). Aeciospores lead to the formation of urediniospores, which drive repeated infections throughout the season. Urediniospores appear as cinnamon-brown pustules (1-2 mm), commonly on lower leaves and are surrounded by a yellow, non-removable halo. They are wind-dispersed, germinate under moist conditions and enter through stomata. Late in the season, the cycle is completed by forming firm black telia (4, 22).

Disease diversity

Four races of cultivated sunflower in Canada were identified using three lines created by the Canadian Department of Agriculture (25). These were named Races 1, 2, 3 and 4. Nearly two thirds of the rust gathered in Manitoba in the 1950s was from Race 1, the least virulent race. The most virulent race at the time, Race 4, made up 2 % of the isolates, followed by Races 2 and 3. Using seven differentials, ten races were categorized in Argentina, which were tried to be related to races in North America (26).

Control measures

Fungicides such as triazoles or strobilurins should be applied at the first sign of disease, especially during periods conducive to rust development. Removing and destroying infected crop debris after harvest can significantly reduce the overwintering inoculum. Crop rotation with non-host plants also helps in breaking the disease cycle (22).

Head rot

Symptoms

Sclerotinia species can cause head rot and basal stem rot at any time, although they usually show symptoms right before flowering. A *sclerotinia* infection swiftly kills the plants, causing the crop to fail. Internally colonized, the fungus eats away at the internal tissues. During flowering, the fungus typically infects the back of the head. The tissues turn pale brown, spongy and squishy as the condition worsens. Eventually, only the fibrous strands at the back of the head and upper stalk remain due to this infection, which also spreads into the developing head and down the stalk. The sheer weight of the diseased seeds at the front of the head causes them to finally fall out of the head (4).

Effects and losses

Head rot affects both the yield and quality of seeds by directly producing severe effects on the capitulum. One of the most common diseases affecting sunflowers worldwide is *Sclerotinia* head rot. For *S. sclerotiorum* in particular, complete resistance is not found but there has been a 60 % reduction in susceptibility since 1970 and, as no isolate-variety interactions exist, this resistance should be durable (4).

Disease cycle

Sclerotinia sclerotiorum, the fungus causing head rot in sunflower, survives in soil as hard, black resting bodies called sclerotia. Under cool, moist conditions, especially during flowering, sclerotia germinate to form small mushroom-like structures (apothecia) that release airborne ascospores. These

spores land on sunflower heads, especially on senescing tissues or wounds and germinate in the presence of moisture. The fungus invades through florets and spreads through the head tissue, forming soft, white, cottony mycelium and producing new sclerotia inside or on the sunflower head. These sclerotia return to the soil at harvest and can remain viable for many years, repeating the cycle (4).

Control measures

Control measures include crop rotation with cereals or other non-hosts for at least three years to reduce soil borne sclerotia. Avoiding overhead irrigation or excessive moisture during flowering is crucial, as the disease favours humid conditions. Fungicides such as mancozeb or chlorothalonil can be applied. Biological control agents such as *Coniothyrium minitans* may help reduce sclerotia viability. Timely fungicide applications during flowering can also be effective, especially in high-risk weather conditions (22).

Leaf spot

Symptoms

The distinctive feature of *Alternaria* leaf spot disease is the appearance of dark brown to black, round to oval spots on leaves, stems and the calyx of flowers. These spots are surrounded by necrotic chlorotic zones with a gray-white necrotic center surrounded by concentric rings and a yellow halo (4). Severe infections cause lesions to coalesce and become irregular, which causes blight and the plant's mortality.

Effects and losses

In India, yield losses range from 27 % to 80 % because of sunflower leaf spot and affect the seed quality and its germination rate (4).

Disease cycle

Alternaria helianthi, the causal agent of sunflower leaf spot, survives in infected plant debris or seeds as conidia or mycelium. In favourable conditions (warm temperatures and high humidity) the conidia are wind- or rain-splashed onto sunflower foliage. The spores germinate and directly penetrate leaf tissue, forming lesions that expand and produce concentric rings. The fungus sporulates again from these lesions, releasing more conidia for secondary infections. Repeated cycles during the season can lead to severe defoliation, especially under prolonged wet conditions. Infected residues left in the field or infected seeds serve as the primary source of inoculum for the next season (4).

Control measures

Foliar fungicides such as mancozeb or chlorothalonil should be applied when symptoms first appear. Rotating with non-host crops and removing infected plant debris at the end of the season are essential practices to reduce inoculum carryover and disease incidence in subsequent seasons (22).

Necrosis

Symptoms

The characteristic field symptoms include mosaic pattern, extensive necrosis of leaf lamina, petiole, stem, floral calyx and complete death of plants ultimately. Early infection either kills or causes serve stunting of plants, malformation of heads and chaffy seeds. Necrosis at bud stage makes the head to bend and twist and results in complete failure of seed setting.

Necrosis leads to complete death of the plant by affecting all the parts of the plant (27).

Effects and losses

The majority of hybrid sunflowers being grown in India have demonstrated varying degrees of disease vulnerability for sunflower necrosis (27). The production of sunflower has been practically threatened in later years by outbreaks of this disease in key sunflower-growing states of India, particularly Andhra, Karnataka and Maharashtra. Reports of yield losses ranging from 30 % to 100 % have been made (28).

Disease cycle

Sunflower necrosis is observed at all growth stages initiating from seedlings to harvest. The disease is caused by tobacco streak virus (TSV) (29) and it was found to be transmitted by thrips (30). The condition is caused by a virus, making it difficult to treat with a single strategy. Tobacco streak virus (TSV), which causes necrosis in sunflower, is primarily transmitted by thrips in a non-persistent manner, meaning the insects acquire and transmit the virus quickly while feeding on infected pollen or plant sap (31). Once inside the plant, TSV spreads systemically through the vascular tissues, leading to symptoms such as necrotic lesions on leaves, stems and flower heads, stunted growth and complete plant death. Thrips moving between infected weeds and sunflowers help spread the virus during the growing season. Weed hosts and volunteer sunflower plants often act as reservoirs of the virus between cropping cycles (30).

Control measures

Necrosis is mainly spread by thrips, hence vector control is crucial. This includes monitoring and reducing thrips populations using systemic insecticides, such as imidacloprid which can be effective against thrips that burrow into plant tissue. Avoiding the proximity of sunflower fields to alternate host crops like cotton and groundnut can reduce the risk of virus spread. Using virus-free seeds and resistant hybrids, as well as maintaining field hygiene.

Majority of the sunflower production worldwide is affected by these diseases and hence development of resistant lines is essential to improve the efficient sunflower cultivation.

Breeding strategies to improve disease resistance

Breeding strategies for disease resistance in sunflower (*Helianthus annuus* L.) have undergone significant advancement, integrating classical methods with modern genomic tools. Initially, conventional breeding relied on phenotypic selection focusing on visual assessment of disease symptoms in field trials. From this a breakthrough came with the utilization of wild *Helianthus* species, which serve as a rich source of resistance genes. For example, *Helianthus argophyllus* and *H. tuberosus* have been exploited to introgress resistance genes against *Plasmopara halstedii* and *Sclerotinia sclerotiorum* (32). This method, though foundational, was limited by environmental influence and the time consumed to develop resistant lines by conventional breeding is longer. Hence, scientists adopted molecular breeding techniques to overcome these limitations. The development of quantitative trait loci (QTLs) has significantly improved our understanding of disease resistance genetics. For instance, the identification of *Pl* resistance genes from wild relatives has played a vital role in downy mildew management. Coupled with MAS, the

application of molecular markers such as SSRs, SNPs and In/Dels has enabled breeders to select for resistance alleles with greater precision and efficiency (33-35). To enhance mapping resolution and dissect complex traits, multi-parent advanced generation inter-cross (MAGIC) populations have been developed. These populations capture greater allelic diversity and recombination events compared to biparental crosses (36).

Furthermore, omics approaches including genomics, transcriptomics, proteomics and metabolomics have deepened our insights into the molecular mechanisms of resistance. Proteomic profiling has highlighted stress-responsive proteins, while metabolomics has uncovered phytohormones associated with resistance responses (37). Advancements in next-generation sequencing (NGS) and genome-wide association studies (GWAS) have further accelerated the identification of candidate genes across diverse germplasm collections. Recently, gene editing tools such as CRISPR/Cas9 have been proposed for precise manipulation of resistance genes, although their application in sunflower is still in early stages. Nonetheless, these combined strategies are shaping a new era of precision breeding, aiming for durable, broad-spectrum resistance against evolving pathogen threats in sunflower (38). Various breeding strategies employed in sunflower are depicted in Fig. 2.

Resistance mechanisms offered by host

It has been found that INA (dichloroisonicotinic acid) and BABA (β -aminobutyric acid) might cause systemic resistance to downy mildew in sunflower, also BTH (benzo(1,2,3)-thiadiazole-7-carbothioic acid S-methyl ester) could cause resistance. According to findings, induced resistance is a practical strategy for managing sunflower downy mildew (39).

The antioxidant enzyme activities of resistant genotypes are substantially higher than those of susceptible genotypes when it comes to host defense response mechanisms against powdery mildew in sunflower. Transcripts of *NPR1* gene, the master regulator of the salicylic acid-based resistance signalling that contributes to plants' resistance against biotrophic pathogens and *hsr203J* gene, which has been implicated in cell death, were found in greater amounts in the resistant genotypes (40).

It was discovered that pathogen infection in sunflower changed the activities of several antioxidant enzymes. Furthermore, head rot (*S. sclerotiorum*) infection significantly increased the concentration of two stress-inducible phytohormones, such as salicylic acid and abscisic acid. Similarly, after infection, the resistant plants' leaves had considerably higher levels of total protein, carbohydrates and chlorogenic acid than the susceptible plants', suggesting a positive relationship between resistance and these suggestive indicators (41).

Resistant genes identified for major disease resistance

Understanding the genetic basis of disease resistance in sunflower is the key to effective breeding strategies. Resistance genes in sunflower can be identified using a range of genetic and biotechnological methods. One of the most widely used approaches is QTL mapping, which involves crossing resistant and susceptible genotypes to develop mapping populations, followed by phenotyping for disease response and genotyping using molecular markers to locate genomic regions associated

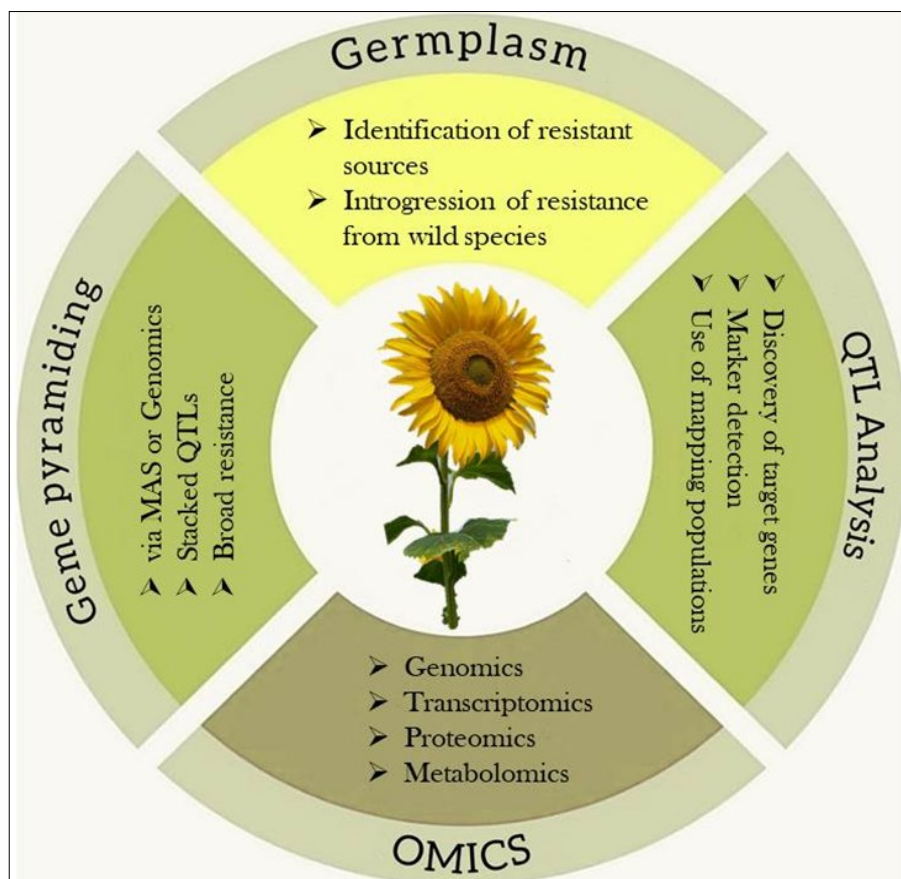


Fig. 2. Breeding strategies and tools for disease resistance in sunflower.

with resistance traits. GWAS offer another powerful tool, particularly for traits controlled by multiple genes, by examining natural populations to establish associations between phenotypic variation and specific genetic markers such as single nucleotide polymorphisms (SNPs) (38). Comparative genomics further aids in resistance gene identification by analyzing conserved regions between resistant and susceptible genotypes or related species. Transcriptomics, particularly RNA sequencing (RNA-Seq), provides insights into genes that are differentially expressed during pathogen attack, helping to pinpoint those involved in defense responses (42). Additionally, mutagenesis followed by Targeting Induced Local Lesions IN Genomes (TILLING) can be used to generate and identify functional mutations in candidate genes (43). Functional validation of resistance genes is often achieved through genome editing tools such as CRISPR-Cas, allowing direct assessment of gene function. Once identified, these resistance genes can be efficiently incorporated into elite cultivars using marker-assisted selection or marker-assisted backcross breeding and even pyramided to develop broad-spectrum and durable disease resistance in sunflower.

Several resistance genes have been mapped to specific linkage groups in the sunflower genome. Molecular markers linked to these genes aid in precise and efficient selection of genotypes during breeding process. For an instance, in case of downy mildew resistance, nearly 27 resistant genes have been identified and majority of them are associated with linkage group 8 and 13 (44). Similarly powdery mildew resistant genes were identified in linkage group 10 (9, 23) and in head rot resistance, 16 genomic regions were majorly identified in 8 chromosomes (45). These genetic tools are essential for developing resistant varieties through marker-assisted

selection. Various genes conferring resistance against major diseases along with their linkage group and markers associated with them are mentioned in Table 2.

Resistant sources for major disease resistance in wild populations

Wild species of sunflower possess diverse traits that are often absent in cultivated varieties. Hence, they serve as valuable genetic resources for improving disease resistance in advanced sunflower varieties/hybrids. Identifying these species helps in broadening the genetic base of sunflower breeding programs. Among various wild *Helianthus* spp., *Helianthus argophyllus* (46) and *Helianthus debilis* (47) contribute resistance to majority of the diseases such as downy mildew, powdery mildew, rust and were widely utilised in pre-breeding programme by possessing relatively good compatibility. Various wild species possessing resistance against major diseases are listed in Table 3.

Molecular markers and mapping approaches

Using RAPD markers, the first map of wild sunflower was created during 1993 (48). A few years later, maps were created and released utilizing RFLP markers that were not based on PCR in various cultivated sunflower crosses (49).

The maps were then updated to include AFLP markers (50). The number of haploid chromosomes in sunflowers is represented by the 17 linkage groups (LG) found in most sunflower linkage maps. Genetic maps based on SSR markers came after these maps (51).

The first composite genetic SSR map covered 1423 cM and included 379 public and proprietary markers in addition to 278 single-locus SSR markers. In order to investigate three new

Table 2. Genes conferring resistance against major diseases of sunflower

SI. No	Linkage group	Genes identified	Markers associated	Reference
Downy Mildew				
1.	LG1	<i>Pl35</i>	NSA_006938, NSA_005423, NSA_009758 & NSA_000630,	(76)
		<i>Pl arg</i>	NSA_007595, NSA_001835	(74)
			ORS-316, ORS-630	(44)
2.	LG2	<i>Pl5</i>	CRT214 & ORS203	(70)
		<i>Pl18</i>	SFW03013 & SFW03060	(76)
			ORS963 & HT298	
3.	LG4	<i>Pl19</i>	ORS644, ORS963, SFW04901 & SFW04052	(71)
		<i>Pl33</i>	NSA_006089, NSA_008496	
			SFW02745, SFW09076, S8_11272025,	
		<i>Pl20</i>	S8_11272046	(74)
		<i>Pl20</i>	SFW01920 & S8_100385559	(76)
4.	LG 8	<i>Pl6</i>	ORS328	(66)
		<i>Pl8</i>	ORS781	(44)
		<i>Pl1</i>	SCT06 (950 b)	(69)
		<i>Pl15</i>	SFW01920, SFW00128, SFW05824 NSA_008457	
			ORS-333	(44)
5.	LG9	<i>Pl2</i>	SFW-00211 & SFW-01272	(65)
6.	LG11	<i>Pl12</i>	CRT-275 & ZVG-53	(68)
		<i>Pl13a, Pl13b</i>	ORS-316	
		<i>Pl4</i>	ORS-316	
		<i>Pl4u</i>	ORS-799, ORS-45	(44)
		<i>Pl11</i>	ORS-728 & ORS-45	(67)
7.	LG13	<i>Pl har6</i>	ZVG-61 & ORS-581	(75)
		<i>Pl arg</i>	ORS662, ORS509, ORS-316, NSA-001392, NSA-002798	
		<i>Pl8</i>	SFW01497, SFW06597	
		<i>Pl8</i>	SFW05743	
Powdery Mildew				
8.	LG 5		ORS 1028, ORS 538	(23)
9.	LG 10		ORS 691, ORS 853, ORS 78	(23)
			ORS 1110, ORS 684	(9)
Rust				
10.	LG 2	<i>R5</i>	SFW01272, NSA_007071, NSA_001605, SFW03654	(67)
11.	LG 8	<i>R1</i>		(65)
		<i>R15</i>		(69)
		<i>R12</i>		(79)
12.	LG 11	<i>R14</i>		(80)
		<i>R4</i>		
		<i>R3</i>		(65)
		<i>R2/R10</i>		(67)
		<i>R11</i>		(71)
		<i>R13a</i>		(77)
13.	LG 13	<i>R13b</i>	NSA_000267, ORS316, ZVG61, SFW05240, HT382	(78)
		<i>R16</i>		(79)
		<i>Pu6</i>		(81)
		<i>Radv</i>		(82)
		<i>R17</i>		
		<i>R18</i>		
14.	LG 14	<i>R2</i>	HT567, SFW00211, SFW01272	(65)
Head Rot				
15.	LG 1	<i>Qhr-1.1</i>	C1_4397272, C1_4397312, C1_4777966	
		<i>Qhr-1.2</i>	C1_52669431, C1_52709188, C1_53853108, C1_58607030	
		<i>Qhr-1.3</i>	C1_77649943, C1_76872191, C1_77560891, C1_85024024	
16.	LG 2	<i>Qhr-2.1</i>	C2_13204329, C2_23136760	
17.	LG 10	<i>Qhr-10.1</i>	C10_134531054, C10_138804279	
18.	LG 12	<i>Qhr-12.1</i>	C12_25882328, C12_25210925, C12_27633722, C12_29909279	
19.	LG 13	<i>Qhr-13.1</i>	C13_163298005, C13_164438892, C13_164412093	(45)
20.	LG 14	<i>Qhr-14.1</i>	C14_28826540, C14_28826567	
		<i>Qhr-14.2</i>	C14_131942451, C14_131764128	
21.	LG 16	<i>Qhr-16.1</i>	C16_21796525, C16_23076152, C16_23084558	
		<i>Qhr-16.2</i>	C16_200693781, C16_201545537	
		<i>Qhr-17.1</i>	C17_3382790, C17_4443495	
		<i>Qhr-17.2</i>	C17_17987938, C17_18206805	
22.	LG 17	<i>Qhr-17.3</i>	C17_128483584, C17_136177433	
		<i>Qhr-17.4</i>	C17_147584722, C17_149360561	
		<i>Qhr-17.5</i>	C17_169747479, C17_191024502	

mapping populations, this map which is currently used as the standard genetic map for sunflower (52) was further saturated with more SSR markers (51).

More than 2000 SSR markers are currently available for mapping and genotyping after being derived from genomic sequences (gSSR) and EST (EST-SSR) (53-56). Ultimately, SNP-based markers enabled the transition to high-density maps. Next-generation sequencing technology enabled whole genome sequencing and genotype-by-sequencing, which enabled genomic selection and GWAS in sunflower and the fabrication of vast quantities of SNP markers for high density maps and SNP arrays. This SNP-based map was used to fine-map the rust resistance gene *R12*. To further address and orient the linkage groupings in accordance with the sunflower reference genetic map, 118 SSR markers were added to the SNP map (37).

Forty-two SSR and/or SNP markers that were found using a 384 Illumina SNP-oligo pool array were used to compare the two marker types' estimated and observed heterozygosity as well as clustering using STRUCTURE and Discriminant Analysis of Principle Components (DAPC). The maintainer/restorer characteristic dominated the population structure, as in previous investigations (57, 58).

The newly created high-density maps in sunflower have a high resolution, which makes it easier to identify and clone genes for a variety of pertinent features in the future for the regions of interest. Furthermore, SNP-based maps provide markers that are closely associated with, for example, resistance genes. These markers can be used in extensive marker-assisted breeding initiatives or included into SNP arrays.

Genomic selection

For association mapping two approaches have been explored:

- (1) Genome-wide association studies (GWAS) and
- (2) Candidate gene approaches.

Genomic selection (GS) selects the individuals based on genomic breeding values (GEBVs) (38). Establishing a genotyped and phenotyped training population is the initial stage in GS. Three key distinctions between GS and regular MAS are readily apparent:

- (1) MAS identifies markers associated with a gene of interest and

quantitative traits during the training phase, while GS models are created to predict GEBVs,

- (2) Traditional MAS uses only a small number of markers for genotyping during the breeding phase, while GS gathers genome-wide data and
- (3) Traditional MAS uses only the identified markers to select individuals by genotype during the breeding phase, while GS selection is based on the GEBV (59).

If nothing is known about one or both parental lines, GS can predict the unknown parental GCA by using the model trained using both phenotyped and genotyped hybrids and can significantly increase breeding efficiency when compared to traditional GCA modelling (60).

The introduction of monogenic characteristics, particularly disease and herbicide resistance, into the breeding material has been accomplished with success using traditional MAS. The limitations of using identified molecular markers in different genetic backgrounds have also been demonstrated by validation of these markers across genotypes. Regarding disease responses of susceptible and resistant genotypes to pathogens such as *Plasmopara halstedii* (61), *S. sclerotiorum* (62) and *Verticillium dahlia* (63), transcriptional profiling has been carried out.

RNASeq or microarray studies have been used to examine the response of sunflower genotypes to drought using transcriptome analysis in water-limited environments compared to well-watered plants (42).

Sunflower has been mutagenized in order to enhance the genetic heterogeneity that is naturally present (64). Recently a TILLING (Targeted Induced Local Lesion In Genomes) population was created for high throughput screening of EMS (ethyl methane sulfonate)-induced mutations in sunflower, which is utilized for research on genes related in the synthesis of fatty acids (43).

A solid foundation for the localization and mapping of just inherited traits was established by developed linkage maps. Due to their dominant inheritance, the majority of the downy mildew resistance genes that confer resistance to the oomycete *Plasmopara halstedii* are reasonably easy to map using molecular markers. Finding markers that are closely related serves as a solid foundation for map-based gene cloning.

Table 3. Wild species possessing resistance against major diseases of sunflower

SI. No	Source of resistance	Reference
Downy Mildew		
1.	<i>H. annuus</i> , <i>H. petiolaris</i> , <i>H. tuberosus</i> and <i>H. praecox</i> ssp. <i>runyonii</i> , <i>H. argophyllus</i> , <i>H. pumilus</i> , <i>H. salicifolius</i>	(46, 83-86)
Powdery Mildew		
2.	<i>H. agrestis</i> , <i>H. angustifolius</i> , <i>H. annuus</i> , <i>H. argophyllus</i> , <i>H. atrorubens</i> , <i>H. bolanderi</i> , <i>H. californicus</i> , <i>H. ciliaris</i> , <i>H. debilis</i> ssp. <i>debilis</i> , <i>H. debilis</i> ssp. <i>vestitus</i> , <i>H. decapetalus</i> , <i>H. divaricatus</i> , <i>H. eggertii</i> , <i>H. giganteus</i> , <i>H. glaucophyllus</i> , <i>H. hirsutus</i> , <i>H. laciniatus</i> , <i>H. laetiflorus</i> , <i>H. laevigatus</i> , <i>H. microcephalus</i> , <i>H. multiflorus</i> , <i>H. pauciflorus</i> , <i>H. praecox</i> subsp. <i>praecox</i> , <i>H. resinosus</i> , <i>H. rigidus</i> , <i>H. salicifolius</i> , <i>H. silphoides</i> , <i>H. simulans</i> , <i>H. smithii</i> , <i>H. strumosus</i> , <i>H. tuberosus</i>	(16, 47, 87-91)
Rust		
3.	<i>H. annuus</i> , <i>H. argophyllus</i> , <i>H. grosseserratus</i> , <i>H. maximiliani</i> , <i>H. nuttallii</i> , <i>H. pauciflorus</i> , <i>H. petiolaris</i> , <i>H. tuberosus</i>	(16, 67, 92)
Head Rot		
4.	<i>Helianthus angustifolius</i> , <i>H. argophyllus</i> , <i>H. californicus</i> , <i>H. debilis</i> , <i>H. decapetalus</i> , <i>H. divaricatus</i> , <i>H. giganteus</i> , <i>H. grosseserratus</i> , <i>H. maximiliani</i> , <i>H. neglectus</i> , <i>H. niveus</i> , <i>H. nuttallii</i> , <i>H. petiolaris</i> , <i>H. praecox</i> ssp. <i>Runyonii</i> , <i>H. resinosus</i> , <i>H. salicifolius</i> , <i>H. strumosus</i> , <i>H. tuberosus</i>	(93-98)
Leaf Spot		
5.	<i>H. debilis</i> , <i>H. decapetalus</i> , <i>H. divaricatus</i> , <i>H. grosseserratus</i> , <i>H. hirsutus</i> , <i>H. mollis</i> , <i>H. maximiliani</i> , <i>H. occidentalis</i> , <i>H. pauciflorus</i> , <i>H. resinosus</i> , <i>H. simulans</i> , <i>H. strumosus</i> , <i>H. tuberosus</i>	(99)

The new high-throughput technologies combined with new genomic-based breeding strategies give us the opportunity, as never before, to understand and mine genetic variation and to use it for improvement of sunflower cultivars.

Conclusion

Genetic studies on major disease resistance in sunflower advances our understanding in the areas of complex interactions between host genome and pathogen. Conventional breeding has been crucial to the development of new sunflower varieties and hybrids over decades. However, the selection process and the difficulty in choosing appropriate genotypes owing to the quantitative nature of traits pose significant challenges like lengthy breeding procedures, mis-prediction, mis-selection, unfavourable environment etc. This has led breeders to increasingly adopt molecular technology (molecular breeding) to enhance precision and efficiency. In molecular breeding, the development of QTL mapping and association mapping facilitate the development of molecular markers that can be utilised in marker assisted selection in resistance breeding programme. Further development was achieved with the help of omics. This enables the successful introgression of resistant genes from wild species into high yielding cultivars. Although, advanced research in identification and validation of resistant QTLs by combining biotechnological tools and breeding approaches on resistant germplasm pools can be carried out to accelerate the development of broad-spectrum disease resistant and high yielding cultivars in sunflower.

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Authors' contributions

VP involved in concept and writing of the review article; RS helped in concept and idea; RK arranged resources; LR and ES has contributed in editing the manuscript. All the authors were read and approved the final manuscript.

Compliance with ethical standards

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